[HIGH COURT OF AUSTRALIA.]

ADELAIDE STEVEDORING COMPANY LIMITED . . . . . APPELLANT;
RESPONDENT,

AND

FORST . . . . . RESPONDENT.
APPLICANT,

ON APPEAL FROM THE SUPREME COURT OF SOUTH AUSTRALIA.

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ADELAIDE, *Sept.* 24.

MELBOURNE, *Dec.* 19.

Rich A.C.J., Starke, Dixon and McTiernan JJ.

Workers' Compensation—Accident arising out of and in the course of employment—Death of workman following exertion—Death due to coronary thrombosis—Conflict of medical evidence as to relation of death to employment—Workmen's Compensation Act 1932-1935 (*S.A.*) (*No.* 2103—*No.* 2246), *sec.* 4.

A waterside worker and winchman was engaged in discharging cargo from a liner. He was a powerful and vigorous man, and, until his collapse, on the day of his death, he appeared to be in normal health. On that day, after performing two tasks which involved muscular exertion, he collapsed and shortly afterwards died. On an application by his widow for compensation under the *Workmen's Compensation Act* 1932-1935 (S.A.), the arbitrator, after hearing medical evidence which disclosed a conflict of opinion, found that the workman's death was due to coronary thrombosis, but that death from coronary thrombosis could not generally be related to exertion and therefore that the workman's death could not satisfactorily be said to have been caused by his exertion. He accordingly found that the death was not the result of injury by accident arising out of and in the course of the employment within the meaning of sec. 4 of the Act. On appeal the Supreme Court (which, under sec. 41 of the Act, had power to rehear the case and to decide questions of fact as well as of law) decided that, although the expert evidence was not conclusive, it showed that physical exertion was commonly, though not invariably, the inciting cause of coronary thrombosis, and that, having regard to the probabilities, the proper conclusion was that the workman's exertion was in fact the cause of it in this case. The court therefore set aside the finding of

the arbitrator and made an affirmative finding that the workman's death was the result of an injury by accident arising out of and in the course of his employment.

Held, by *Rich* A.C.J., *Starke* and *McTiernan* JJ. (*Dixon* J. dissenting), that on the evidence the finding of the Supreme Court was justified and should not be disturbed.

Decision of the Supreme Court of South Australia (Full Court) affirmed.

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APPEAL from the Supreme Court of South Australia.

Carl Heinrich Meyer Forst was employed as a workman (within the meaning of that term in the *Workmen's Compensation Act* 1932-1935 (S.A.)) by the Adelaide Stevedoring Co. Ltd. He was a powerful man, weighing fifteen to sixteen stone, and was employed as a waterside worker and winchman. He was sixty-three years of age. On 4th June 1938 he was engaged in discharging cargo from the s.s. *Maloja*. He started work at 8 a.m., and, throughout the day, until his death, which occurred shortly after 4 p.m., he appeared to be in normal health. About 4 p.m. the wire ran off one of the sheaves of the crane which he was operating. The crane was then at an angle of about forty-five degrees, and Forst climbed up the lattice-work of the crane and attempted to replace the wire. In this attempt he lay prone on the crane and, taking hold of the wire, tried to lift it and push it over the gin. This task required muscular exertion which he was in a difficult position to perform. After two or three unsuccessful attempts he descended and gave every appearance of being quite normal. His reason for coming down was that he found that the job was going to be difficult and he decided to leave it to the crew, whose duty, strictly, it was. His foreman then instructed him to assist to pull down the wire of another crane. He wheeled round, apparently quite normally, and began that job, which involved his pulling the wire with his hands over his head. He was obliged to pull down each hand alternately, until it was somewhere near the level of his face. This work, also, was not easy and needed effort. Another workman, whom he was assisting in this task and who was working face to face with him, noticed nothing wrong with him. When the pulling was to all intents and purposes completed, Forst walked away. After walking about twelve yards he collapsed, lost consciousness and died within a very short time.

His widow, Amy Forst, claimed from the company compensation under the *Workmen's Compensation Act* 1932-1935 (S.A.).

The question whether the work done by Forst, and in particular the exertion which he expended, contributed in any material degree to the condition from which his death arose was investigated first

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by a special magistrate acting as arbitrator under the *Workmen's Compensation Act* 1932-1935. Medical witnesses were called, all of whom agreed that Forst had died of cardiac insufficiency, but they differed in opinion as to whether coronary thrombosis was the cause, and, if so, whether it had any relation to the exertion which he had undergone. The medical evidence was substantially as appears hereunder. (All the medical witnesses, except Dr. Gartrell and Professor Cleland, were called by the applicant; but the evidence of Dr. Gartrell was interposed by leave at the stage indicated by the order in which the evidence of the witnesses is set out hereunder.)

Dr. Cherry :—

" On the 6th June 1938 I was requested to conduct a post-mortem examination of the body of Mr. Forst. I saw the body at the Outer Harbour in a shed adjoining a wharf. The body was lying on a hatch fully clothed. The body was dead but warm and had been dead apparently only a short while. There was a cut on the back of the left side of the head which was wet with recent blood. I saw no other external marks of violence. I was unable then to state the cause of death. I saw the body on Saturday 4th at 4.40 p.m. On Monday 6th June at the Port Adelaide Morgue I performed a post-mortem examination on the same body. That examination was done at the coroner's request. On the back of the left side of the head was a ragged cut about 1 inch long extending to the bone. Blood was evident on the scalp around this wound. There was no sign of fracture of the skull nor of internal injury to the brain nor was there any sign of cerebral haemorrhage. The heart was enlarged and showed signs of chronic valvular disease. There was atheroma of the aorta. The anterior coronary artery was blocked with a clot. This clot was not confined to the lumen. The lumen is the space of the artery. The clot was not confined to the lumen of the artery but extended through the inner lining of the vessel of the coronary artery into its wall. The other organs of the body were in a healthy condition and there were no other marks of violence. Death in my opinion was due to thrombosis of the coronary artery and in my opinion was sudden. Thrombosis means clotting of blood on its site. I could find no other cause of death. . . . The heart was generally enlarged especially the left ventricle. The clot that I found proceeded from an atheromatous patch. Atheroma is a fibrous disease of the inner lining of arteries which destroys their elasticity and normal distension of the vessel. Having regard to the pathological condition that I found in the coronary artery I would say that had I known that the man was suffering from symptoms and signs indicating coronary disease I

would advise him against heavy work, because any undue strain would tend to cause dilatation and rupture of a diseased artery or the separation of any clot which would cause an embolus. Embolus is a clot that shifts from its site of origin to another spot and blocks another vessel. The rupture that I spoke of would be at the point of the disease. The atheromatous patch would cause a small aneurismal dilatation. An aneurism is a swelling due to the internal pressure. There is a suggestion of aneurism in this case. This aneurismal dilatation is not what we call an aneurism. There was no microscopic examination. I did not find it necessary to measure the auricle and ventricle. I have no recollection of the quantity of blood that was at the heart or other organs. . . . The reason that I did not measure the auricles and ventricles was that I saw no reason and it did not occur to me that it was necessary to measure them. The cause of death was quite clear. Dr. E. F. Gartrell was with me at the time that I conducted the post-mortem. It is generally recognized that thrombosis is not a condition that is generally brought about by exertion. In this case I do not say that probably his work had nothing to do with the thrombosis that I discovered. The probabilities are that the thrombosis would have formed as it in fact did form at the time it did form whether he had been working or not. It might have occurred in his sleep. Thrombosis does often occur in sleep. The inner wall of the vessel was ruptured and the clot extended beyond the inner wall of the vessel. In my opinion that was less likely to occur at rest. The ruptured wall was at the site of some atheroma. The spreading of the clot had gone under the flaky condition. The flakes do not separate. It had gone under that flaky condition in this case. . . . That is not usual in thrombosis. If you get a blood pressure forces the blood under that weak spot. . . . That is from my reading and from witnessing post-mortems. Thrombosis is a fairly common cause of sudden death in heart cases. Although I say that the blood had spread under the atheroma I think that the clot was not very long in forming. I would not express any opinion as to how long it took to form. If I had known his condition I would have advised him against heavy work for the reasons that I have given. They are the only reasons. He was suffering from signs and symptoms of heart breathlessness on exertion and pain in the heart. When I say that it might have caused a rupture of the diseased arteries and an embolus I would not say that those are not the only conditions that I would advise him against hard work for. He might have other dilatations of the heart or dropsy which would not be immediately fatal. There was a distinct thrombosis, it was not

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an embolus, and that is why the blood got in under the wall. The clot might have been an embolus. In this case in my opinion it was not an embolus but was a thrombus. There was nothing else that could have suggested exertion but the fracture. It was the atheroma that caused the rupture. It predisposed the rupture. It was a weak spot. The atheromatous condition caused the clot to form on the ruptured patch. . . . Exertion could not cause the clot to form on the inner side. When once a thing was ruptured the clot would form on the other side. The exertion would not cause the rupture to form on the inner side, that is, inside the vessel. That is the inner side of the atheromatous patch but the clot forming on the outer side on the atheromatous patch would be due to blood coming through the wall of the vessel and this passage through the wall of the vessel would be permitted by rupture at the side of the patch and consequent passage of blood from the vessel into its wall. In this case rupture of intima may or may not have been caused by exertion. . . . This man had two thrombi, one inside and one out, but contiguous. Thrombus is not really a thrombus if it is outside the vessel. I call it a secondary thrombus. It was not sufficiently to form an aneurism on the outside of the vessel. It was a seepage through. . . . These two thrombi that I referred to were one clot extended through the intima, which is the inner lining of the artery. . . . It could have occurred as a result of exertion. . . . It is more likely to occur after exertion. . . . I did not say that the primary clot was the cause of death. The secondary clot was increasing in size and it could bulge through the wall of the vessel. The thing would be more or less continuous. . . . The cause of the death was due to both primary and secondary thrombosis. . . . The seepage through would distend the wall of the vessel and the distention would be more towards the lumina of the vessel than the other so that you would get your blockage. I am unable to state whether the atheromatous condition did not first cause the clot to form in the lumen and then there was a seepage through under the intima. In this particular case it is impossible to say which occurred first. By primary clot I mean the first clot and that is the one inside."

Dr. Covernton :—

"I knew the late Mr. Forst. I attended to him at various times. I remember attending to him in June 1937. I was sent for to see him in the later afternoon. He had been brought home from the wharf. He was suffering from shock collapse and severe pain. I was told that he had had a severe attack of vomiting on the wharf and that he was brought home immediately afterward. At

that time when I first saw him I thought that he might be suffering from a severe abdominal condition possibly a perforating ulcer of the stomach but the symptoms improved and I saw him the next day when he was very much better, said that he would be able to go to work in the afternoon. When I saw him the next day I thought that the symptoms pointed more to the possibility of there being a coronary thrombosis. He returned to work soon afterwards and I did not see him again until I heard of his death. Having regard to what the post-mortem examination showed I would say that that confirmed my opinion. The effect of sudden exertion on a person suffering from thrombosis of the coronary artery I would say that an exertion might bring on an attack of coronary thrombosis. I think that is the general view of the medical profession. . . . I think that coronary thrombosis may be brought on by exertion. Thrombosis does occur when people are asleep. I would not agree with Dr. Cherry when he says that thrombosis does often occur in sleep. The opinion that thrombosis generally occurs during rest I do not agree with. I have not read of it that thrombosis does generally occur during rest. I have read and heard that thrombosis does occur during rest but I won't say that it often occurs during rest. Thrombosis is the formation of a clot. . . . The artery condition is more favourable to the formation of a clot when it is flowing slowly than when it is being pumped quickly. . . . I think it depends on the nature of the exertion which he is undergoing whether it occurs during rest or during exertion. I mean by that the quantity of exertion and not the actual nature of the exertion. I do not say that if a man had a lot of exertion it is not likely that a thrombus would form. . . . Any exertion which includes the straining and hold the breath slows the circulation through the heart. I mean any exertion. I think it necessitates holding your breath and straining. I should think the exertion of running round a tennis court would be sufficient. Any type of exertion is the exertion I have got in mind for bringing about the circulation. I mean the exertion I have in mind is an exertion which necessitates the closing of the mouth and holding of the breath. It is more particularly with a man lying down and something pressed into his body. For instance a man lying across a log."

Dr. Thiersch :—

"I read of what Forst was doing shortly before he died. I think the cause of death was acute insufficiency of his heart due to strain, that is physical strain. The heart had atheroma of the coronary tissues. The ramus descendens of the left coronary artery is a vital spot. A heart in this condition would be very sensitive to strain,

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that is, abnormal or excess strain. A heart with atheromatosis on this particular spot depending on its particular size the heart would work at the limit of its capacity; when I say that it works near the limits of its capacity I mean that in normal conditions it would perform its functions satisfactorily but if put under strain the heart may show acute insufficiency and death may follow immediately or he may not die. . . . I heard of what Forst was doing just before his death. The strain involved by that would be a heavy strain on the heart. From the description of his work I gather that he was not standing up straight but lying partly down on his abdomen. That is lying partly face downward. That would put a higher strain on the action of the heart itself. Working with his hands over his head would put an extra strain on the heart because both positions reduce the blood flow through the lungs. The fact that he was lifting the wire, he would be holding his breath during that time. I would expect some irregularity in breathing. The moment that he stops breathing the diaphragm does not move in the normal way, and the venous congestion would appear. . . . The heart cannot fill itself properly. Another consequence is that air is not taken into the lungs and the blood is not properly oxygenated. The heart must therefore suffer from lack of blood and oxygen. In a normal person that would return to normal after adapting itself. If there is excess strain put on the heart it would show signs of insufficiency. First, the already decreased circulation through the lungs would decrease further more and less blood would come back from the lungs. This blood would contain little oxygen only and the coronary supply of the heart would be so little that the left ventricle might dilate and auricular fibrillation might occur. . . . As soon as this patient would cease to work so very hard an abnormal amount of venous blood would rush to the heart and the very weak heart which did not have enough oxygen would acutely suffer from insufficiency because it had to pump this enormous amount of venous blood through the body. Then again acute insufficiency with fibrillation of the heart may occur and death follow. The blood accumulates and when breathing is resumed the blood goes to the heart and not having sufficient oxygen it is unable to do it. Having heard or read all of the evidence—the exertion done by Forst, in my opinion the cause of death was acute insufficiency of heart and lack of oxygen. This is a direct result of exertion and excess strain put on the heart. In my opinion it was not due to coronary thrombosis. If it was due to that—I would still think that Forst's death resulted from excess strain. . . . If death was caused by thrombosis that thrombosis would have arisen through exertion

because the rise of the blood pressure breaking the wall of the artery. There would be a decreased flow of blood. The slowing of the blood flow through the artery would tend to thrombosis. . . . I don't think that coronary thrombosis is too clearly defined in medical knowledge that it is brought about by exertion. I don't say that it is brought about by exertion. It is more usual not to be brought about by exertion. . . . Since I have been in Adelaide I have had deaths from coronary thrombosis. None of the cases followed exertion. We had a case in the Adelaide Hospital about a fortnight ago where a man died from coronary thrombosis and a post-mortem was carried out. This man died after he went into the water. Swimming would involve some exertion. I think that in this case exertion contributed to his death, that is the case where the man did swimming."

Dr. Gartrell :—

"On the 6th June of this year I was present when a post-mortem examination was performed on Mr. Forst. I actually dissected out the heart after Dr. Cherry had examined it to some extent. From what I saw I arrived at a conclusion as to what Forst had died from. I say it was coronary thrombosis producing acute cardiac insufficiency. The thrombosis was about three-quarters of an inch from the orifice of the coronary artery descending along the front of the heart. The clot not only occupied part of the lumen of the artery but extended through an aperture in the inner lining of the wall being continuous with a further clot situated between the layers of the wall of the coronary artery and underneath a patch of that wall which was affected by atheroma. I formed an opinion as to when that clot had formed. It must have been quite recent, within hours of death, and certainly before death because unless there had been this aperture leading from the lumen of the artery underneath the diseased patch there would not be any blood underneath that layer to form a clot post-mortem. I say that it was impossible to have a post-mortem clot underneath the atheroma. The blood underneath the atheroma was definitely there before death. I consider that the primary factor of death was the atheromatous condition of the wall of the coronary artery because this was a roughened surface on which clot does frequently form and also because there was no evidence of an embolus coming from anywhere else. The clot might have commenced originally underneath the layer and I think it probably did because it is more easy for the clot to extend through from within out that is from within the wall itself out into the blood stream rather than to reverse through from the lumen of the artery into what must have been merely a potential cavity and

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even then one supported by the layers in the wall of coronary artery. In my opinion exertion would (not) " [*sic*, in transcript] " have caused that. I do not know of any method by which exertion could cause it to form either from inside or outside because under the stress of exertion the influences acting on that artery are of two kinds, one actually movement of the whole heart causing a possible alteration in the shape of the vessel, but as the apex of the heart and the base are relatively fixed points during contraction it is inconceivable to me that a movement of the heart is going to have any material effect in altering the shape of the vessel running directly between those two points. And two, the other influence would be a more rapid flow of blood through the coronary artery and this certainly would not be conducive to clotting. A rapid flow of blood would be less likely than a slow flow to be associated with clotting. These two points which I have raised are probably more of theoretical or hypothical interest than of practical importance and I do not believe that exercise of effort does produce coronary thrombosis which occurs so very frequently during sleep, in fact I have been called to quite as many patients suffering from the onset of coronary thrombosis during the night as during the day and hence I consider that as thrombosis can occur and frequently does so when the patient is resting it is obvious that on some occasions it must occur accidentally during the ordinary work of the day and quite irrespective of it. Coronary thrombosis. The figures vary a great deal and I could quote figures on the one hand which appear to show that professional men were much more subject to it than labourers and among recent figures including over 1,000 cases there was not shown to be much difference between labouring and professional work. . . . After that clot had finished forming Forst's death must have occurred within a few minutes because this clot was sufficient to deprive part of the heart of its blood supply and if this had obtained at the time when he was undergoing some heavy strain I think dealing with some tackle in this case a few minutes before the death, he would most certainly have had symptoms at that time and not have gone on with the subsequent task which I believe was pulling on a rope. I say that he would have had extreme pain or breathlessness would be the commonest but sometimes the patient would fall unconscious under those conditions practically instantaneously. I take that there was a heavy strain up on the jib and I think if there was a strain then he would have had symptoms after he had finished the milder occupation otherwise he could not have gone on with the heavier. His clot could not have been formed five minutes before. I think that with a clot of that size he would fall almost

simultaneously with the complete formation of that clot. It is difficult to say just when the actual process starts but the probability is that it occurs very quickly. Most likely it occurred in a very short time. Assuming that there was not very much difference between the two types of work, knowing that after he had done the second he fell on the deck that would not influence my opinion as regards the relationship between the work and the death for this reason that with a clot of that magnitude occluding a large vessel at a spot so near its origin would inevitably produce sudden death even in the absence of any strain. The atheromatous condition would not have any definite relationship with his work. There is no essential difference between the incidence in different classes of men although there might be a slight percentage in labourers having this condition over others. . . . Referring to my answer as to what I considered the primary factor of death I say that the secondary factor of death was coronary thrombosis inasmuch as the diseased condition is coronary sclerosis of an atheromatous type of which coronary thrombosis is one of the recognized complications. The atheromatous condition might lead to a gradual closing down and blocking of the vessel and starvation of the heart muscle. The condition of this man had not got to that stage. . . . I spoke of the clot proceeding through an aperture in the lumina of the heart. I formed that opinion because a series of cases have recently been reported showing that coronary occlusion had occurred solely due to clot formation underneath the intimal lining of the artery without any rupture into the lumina. As that can occur it was my conception that a clot had formed under the intima and broken through a diseased part of the lining wall. The liver was incised and inspected. I inspected it. It was not obviously enlarged, that is by looking at it I would say that it was not enlarged. I gave a report in connection with this post-mortem. Speaking of it from two different points of view as regards the chronological sequence I thought it commenced under the patch but speaking pathologically when one examined the vessel the first thing to be seen was the clot in the lumina which was then found extending into the cavity. I opened the coronary artery with a pair of fine pointed scissors. I regard the clot under the lining and the clot in the lumina were both of the same consistency and apparently one and the same clot. I do not think that the aperture could not " [*sic*] " have been caused by a rupture caused by exertion because for the reasons already stated exertion for practical purposes does not have much direct affect on the wall of the vessel. . . . As this artery lies on and is adherent to the heart muscle it makes whatever movements it does make to more

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or less the same degree every day and therefore any slight difference in the movement occasioned during exertion would have a very slight effect. Exertion affects the beating of the heart in many ways. Under exertion I would not call the heart twisting to any material importance. Under exertion blood pressure may rise very steeply. That would not be a reasonable possibility in a coronary artery like this where the layer which was ruptured is supported by the other and stronger layers. It is a potential cavity supported by the other layers so that the effect of rise in blood pressure would mainly be to press this inner layer even more firmly against its supporting layers. The effect of exertion would in that way ward off a rupture of this patch but other factors come into it. Other factors would be the speed of the blood flow but you can't estimate how much effect that might have. Another would be any gross disease of the outer walls, that might come into it. Portion of this clot was under the intimal lining. Before it became a clot it must have been blood. Blood must have been under the patch. It might have seeped from the small blood vessels supplying the wall of the artery. I did think it did come from that source. Pressure exerted on the area in question. I think that the pressure exerted directly on to the area itself would be greater in the small vessels. I mean the part of the area which actually provided the blood or through which the blood pressure would be much greater in the coronary artery than in the smaller vessels. . . . A diseased condition of the walls of any artery produces a softening which increases until it may be sufficient to allow the blood to seep through. Exertion would not be an important factor in that because it would occur at a certain time irrespective of exertion and if a man could demonstrate feats of strength on one day it was not likely that exertion on the following day would be an important deciding factor. If a man could perform a feat of strength on one day he will go on to such time as that diseased process would reach the stage that I spoke of. I think exertion would not have anything to do with it, it would just come chronologically because pressure in these very small vessels which nourish the wall of the coronary artery is always very low."

Professor Hicks :—

"I am a professor at the Adelaide University, and have studied pathology in New Zealand, England and on the Continent. I have heard and read the evidence in this case. Both sides agree that death was due to cessation of heart action. I noticed that Dr. Gartrell had said that he had been warned and I agree with that. The danger was that he might die from acute cardiac insufficiency

or that insufficiency might occur which would incapacitate him for life. There is a difference of opinion whether the man died from thrombosis at all, and whether if he did, that it was coincidence or that was caused by exertion or that it was due to a defect in the supply to the heart already damaged. We have the evidence of Dr. Covernton that he examined the man twelve months before an attack after which had subsided caused him to think that it might have been a coronary thrombosis. The attack was typical as described by Dr. Covernton as coronary thrombosis. It was a brief attack and he recovered rapidly. But he might quite well have an attack at any subsequent date. The only evidence that we have is the statement that a clot was found in this coronary artery at a post-mortem thirty-six hours after death and that the coronary vessels had atheromatous patches in them. But we have no proof either by word or by objective evidence that we were dealing with a coronary thrombosis. By that I mean the mere description of a thrombosis itself does not convince me nor does the commentary of the coronary vessels convince me that we were dealing with a coronary thrombosis. I am not convinced that it was not an ante-mortem or a post-mortem clot. It might have been but I am not convinced by the evidence. A post-mortem sufficiently carried out could have produced that evidence. The evidence from this post-mortem in that regard is not convincing. . . . In my opinion certain information is missing from the post-mortem of the blood in respect of the chambers of the organ. The state of the circulation of the lungs, and the weight of the liver, as well as a statement concerning the appearance of the wall of the heart in the region supplied by the vessel which is said to have been blocked. . . . Auricular fibrillation could have started a course of events which might have led to death. That might have been exertion in a subject with a medical history such as we have. If there had been auricular fibrillation we would have found blood accumulated in the lungs, and we might have expected to have found an acutely dilated heart if death had been a subsequent event. We don't know what fibrillations about the lungs or much about the heart except that a clot was found and atheromatous patches were observed in the coronary vessels. The state of the liver might have been very relevant to offset insufficiency in this subject. It might have happened on this day and concerning which he made no complaint. I think . . . the opinion that death was due to coronary thrombosis was based on the finding of that clot and . . . other evidence . . . might have led to a . . . different conclusion.

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Atheroma leads to thrombosis not commonly in the way suggested in this instance but by virtue of the diminution of the calibre of the vessels until the blood flowing through them becomes slow. In addition if there is any nervous reflex which reduces the calibre of the vessels still further in this particular instance thrombosis formation has been associated with the atheromatous patch, and no observations have been made on the limitation of the calibre of the vessels by the atheromatous process. I can't conceive that this patch would not be subject to any forces which might detach it. A sudden rise in pressure by expanding the distensible part of the vessel wall will tend to detach a patch from the surface. . . . If the patch were detached I would ascribe it to the steep rise of pressure extending the vessel at the time when Forst undertook the exertion. That is if this is a cause, it comes into the picture, being a rupture following a steep rise in blood pressure. Dr. Gartrell said that the patch was ruptured from the outside and not the inside. I can only say that that is astonishing news to me, that an uncomplicated atheromatous patch —by uncomplicated I mean no arteritis of the vessels—in an uncomplicated case of atheroma I am astonished to learn that such a patch is likely to be detached by breaking from vessels in the media. Atheroma is a disease of the intima and not of the media where these vessels are. And I would add in any text-book on pathology classical pictures of atheroma plates are to be seen and in none I am prepared to state this haemorrhage underneath them show part of the atheromatous process. Between the heart and the media there are two distinct surfaces. They are not related to each other. If the heart becomes loose they remain unaltered. A classical instance of involving the vessels of the media as the vessel wall as a whole is endarteritis such vessels" [*sic*]. "And then the result is an aneurism and a bulge in the vessel and the rupture of the vessel. The pressure of the blood in the vessel is almost negligible. I tried to get some measure of pressure and found that I could not give the court a figure and I came to the conclusion that it is twenty-five per cent less than in the larger vessels. The pressure in the capillaries amounting to one-fiftieth. It could only seep and form a clot provided there was some degenerated change in the media itself. And in this case I know of no such change. It is new to me. I don't accept it as very good evidence. If there were any such degenerative change as lead to bleeding from the vessels of the media it might lead to such a clot as Dr. Gartrell said he found. But in this case it would have been of such an interesting nature there would be all the more reason for keeping the vessel intact for a microscopic

examination. It was only such a degenerative change that would cause bleeding from the media. The day of miracles is perhaps not over. I can't believe that the blood could have connected up with the blood in the main vessel unless there was some mechanical removal of the plate before. I don't think that the bleeding from the vessels in the media pushed the plate off from the main vessel. I think that if there was any bleeding it was caused by a rupture inside caused by a steep rise in blood pressure. It is remarkable that none of the signs of thrombosis were observed at Forst's death. That all of them were not present is quite commonly found and it is possible to have death from coronary thrombosis without any pain which is otherwise a characteristic and general symptom. But that there was no cardinal sign is to my mind an important matter. That is pain of an intense nature and vomiting and evacuation of the bowel, and acute breathlessness which would be seen in the form of a gasping attack and collapse . . . I have perused an article by Dr. *J. C. Paterson* in the *Archives of Pathology* (1936), vol. 22, p. 313. That deals with the subject of a seepage through the capillaries to form a blood clot. . . . This throws a very interesting light on the phenomena of atheroma itself. And the nature of the bleeding is described but it does not arise from the vessels in the walls of the artery. That is what the article says. The article explains how bleeding does take place but it doesn't say how it couldn't arise. It does say how it could arise though and that is rather germane to this point. In my evidence I said that unless there was arteritis I could not conceive any vessels growing in such a way that a haemorrhage could take place and that arteritis leads to destruction of the media and to aneurism and rupture of the vessel. *Paterson* shows that as part of the atheromatous process there is an endarteritis that is an inflammation of the intima with a down growth of capillaries from the lumen of the main vessel. This of course is quite a different proposition and because of the short length of such capillaries and because they arise directly from the lumen of the main vessel the pressure in them is more of the nature of the pressure in the main vessel. Not as in the case of capillaries of the media where the pressure would be vastly less. *Paterson* claims that a combination of this high pressure and lack of support to the small capillaries in the soft atheromatous tissue is the cause of their rupture and he discusses such conditions as rises of pressure as being a possible cause of such haemorrhage. In the cases he studied he finds in approximately fifty per cent each way thrombosis and absence of thrombosis in association with this

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small local intimal haemorrhage, and he indicates that such haemorrhages might be a factor in precipitating thrombosis but that it is not the only factor. My personal comment is that *Paterson's* contribution is very important to the subject of atheroma and of thrombosis but it is not at all productive of the line of argument which we have dealt with before. That is to say we were dealing with before the possibility of pressure in the vessels of capillaries in the media being added to lift a patch. . . . I say that it doesn't support the story of Dr. Gartrell. I think he has got the story wrong. On the contrary this article would rather show how it was possible for blood pressure to raise in the main blood vessel to help in dislodging an atheromatous patch in itself. The capillaries as described by *Paterson* are very small. . . . The theory as advanced by Dr. Gartrell did not appear to me as possible at all—that is the theory of haemorrhage from the walls of the arteries. The theory I previously made remains the same. I feel more decided about the matter having read *Paterson's* article. The capillaries in the media are directly connected with the lumina so that the pressure in the capillaries is very much the same as the pressure in the lumina. If the pressure in the capillaries rose the pressure in the lumina would rise. If the pressure in the lumina rose for some reason the pressure in the capillaries would be directly related to it. . . . I am aware of the particular form of exertion which forced this position. In my opinion that exertion alone could have led up to death. That is without any pathological condition which could have been seen even microscopically. With this man at this age the heart is muscularly capable in a normal person of meeting all sudden and severe demands upon its strength. The mechanism is highly complex but mainly it is the muscle of the heart; with age this muscular capacity necessarily gets less. It is one of the prime evidences of advancing age, that we lose cardiac capacity to cope with sudden severe demands. Any pathological condition superadded merely hastens the process of cardiac incapacity. In the type of exercise which had been preceding this death blood was impeded in its return to the heart by the nature of the muscular effort which is popularly termed a straining effort where the breath is held. That means that for those few beats of the heart during the effort its own blood supply was diminishing, or shall I say its own oxygen supply was diminishing. That is the oxygen supplied by the corpuscles. When the exercise was over these difficulties in the return of blood to the heart were as suddenly removed and heart muscle which had had the handicap of the previous seconds of failing oxygen supply was now asked to deal with all the pent-up blood. This is the very crux

of the matter of testing the capacity of a heart to survive and in a defective heart it can lead to fibrillation of the auricle and even to death but more likely to fibrillation of the auricle and more or less permanent increased cardiac disability. When I said defective heart I meant aged or with any pathological result added it is worse. As an illustration of straining effort I would mention tug of war, straining at stool when very constipated which has even caused death, trying to swing the motor of an automobile when it is cold. These three types of exercise or effort are all associated with holding the breath and lack of other muscular movement which would bring blood back to the heart, and they are dangerous after a certain age. I have read and heard the evidence concerning the clot. I wouldn't like to say now if the clot was pre-mortal or post-mortal not having seen it first. If I had seen it with the naked eye I would have been helped by the attempt to pass probes down various vessels to see whether their lumina were restricted . . . whether there was extensive atheromatous restriction of the vessels because that is a well-known precursor of thrombosis because it slows the circulation of the blood through those vessels and then as regards the microscopic examination it would have proved whether this clot was in fact an ante-mortem clot because the structure of the clot would have been entirely different to a post-mortem clot. They couldn't have been examined properly without microscopic examination but a person skilled in post-mortem examinations . . . could have looked at that clot and had no difficulty at all as an ante-mortem clot is vastly different to a post-mortem clot in appearance. If that appearance had been given in evidence I don't think I would have been here now. Considering the clot was ante-mortem that could have been produced by the exertion that Forst took. . . . I agree with Dr. Gartrell that the condition predisposing the clot is the slowing of the blood through the arteries. As a result of the exertion at any stage there could have been definitely a stage where a combination of slowing and low oxygen content of the blood could have predisposed this. The low oxygen content is also a condition predisposing a blood-clot formation. That could have been in consequence of the exertion. In my opinion assuming that it was ante-mortem clot I think it is ignoring too great a functional disturbance occasioned by this effort to say that the formation of the clot was not due to disturbances produced by that effort. The effort in other words is the most noteworthy fact in relation to this cardiac death on the day of the death. Assuming it was ante-mortem then it was likely that the death did result from coronary thrombosis but it would be a matter of dialectic at this stage but it wouldn't be incorrect. I

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should say in my opinion that exertion definitely could cause coronary thrombosis and *Paterson's* paper gives a far better basis for such a position than the other known factors. This particular exertion would by this version of *Paterson* by breaching the endothelium by this small haemorrhage and at the same time giving a small supply of oxygen for the blood both of which would be predisposing factors. It is noteworthy that *Paterson* is most emphatic on the point that there are other factors and as he is a pathologist it is definite that he is being scientifically correct. There are no other objective symptoms described save the clot and the sudden death. There is nothing to suggest any subjective symptoms. He did not say anything to anyone there that he had a pain in the abdomen or such. He just fell down and died. This savors of dialectic as these are all variable factors and it is striking that there is an absence of any symptom whereas on the other hand there is definite evidence of previous fibrillation. He would have no warning at all and might just fall dead. There are even cases of falling dead without any exertion. There are cases where a patient has been known to be in such a state by a doctor who has attended him. Considering Forst's effort which was a considerable one that is why I think it is a most dramatic fact. In my opinion on the material before me the most probable explanation for Forst's death that whatever was the ultimate cause of cardiac failure in Forst the effort was the predisposing and predetermining factor, otherwise we are dealing with a coincidence and we have to ignore a gross interference with circulatory function which everybody has been a witness to on that day."

Professor Cleland :—

"I am professor of pathology at the University of Adelaide. I have been the professor of pathology at Adelaide for nineteen years. I have read the evidence which has been given in this case up to this morning. I remember the description of Dr. Gartrell as to how he found Forst's heart at the post-mortem. 'A patch of atheroma in the anterior descending branch of the left coronary artery with a blood clot underneath this patch and another thrombus present in the lumen of the artery and situated three-quarters of an inch from its orifice.' Accepting that description I should say that the cause of death to the person whom the post-mortem was made was coronary thrombosis. In my position of professor of pathology I have conducted a large number of post-mortems at Adelaide Hospital. About 3,000 in South Australia. I have found as a result of those post-mortems quite a number where death has been due to coronary thrombosis. Seems to be increasing. In the last

690 post-mortems there have been twenty-one cases of coronary thrombosis, eleven men and ten women but I conducted more post-mortems on men than women. The usual case would be about two to one in favour of men. I have followed up the medical history of some of those bodies on which the post-mortems were carried out. I looked up the clinical notes. Very few patients die at the Adelaide Hospital instantaneously through coronary thrombosis; hence the following summary of sixteen cases does not include any case of instantaneous death. There doesn't seem to be any reason clearly in association with what they were doing. Generally the circumstances under which the acute symptoms develop from persons doing nothing at all—seated at the table—to a man walking round his home where digging in the garden to the case of a man going to work where the symptoms began when getting on his bicycle. One whilst swimming and one whilst walking. The greatest effort is the man who had done some digging and walked round the garden. I think, however, that the one swimming would be the greatest effort. The attacks seem to be fairly evenly distributed. I have seven cases in which presumably the acute symptoms came on between 6 a.m. and p.m. and nine cases where it came on apparently between 6 p.m. and 8 a.m. . . . I can't speak from actual experience as to whether effort plays any part. From my reading I gather that there is no recognizable association with effort. That does agree with the fact that in formation of a thrombus a sluggish circulation through the part where the thrombus is formed is an assisting factor. Atheromatous patches are common over the age of fifty. They become very common at sixty. I suppose most of us over sixty years have got a few patches in our coronary systems. . . . In my opinion the extension of the occurrence of clot in wall is corroborative evidence supporting the view that it was an ante-mortem clot in the lumen." The witness referred to an article on the "Relation of Physical Exertion and Emotion to Precipitation of Coronary Thrombi," by Dr. *J. C. Paterson*, in the *Journal of the American Medical Association* of 11th March 1939,* and, in reference to the passage in the article

*The text of this article was as follows:—"Until quite recently it was generally agreed that excessive physical exertion or emotion was intimately connected with the precipitation of coronary thrombi. *Fitzhugh* and *Hamilton* and *Sproull*, from analyses of the histories of their cases, concluded that coronary occlusion was preceded as a rule by departures from ordinary habits of living. During the last two years, however, some doubt has been expressed regarding this relationship. *Phipps* and later *Master* and his co-workers in statistical surveys have shown that approximately 40 per cent of attacks of coronary thrombosis are initiated while the patient is either asleep or at rest and that in only a small number is the attack immediately related to unusual exertion or emotion. The latter authors conclude that their results seem to eliminate exertion or excitement as factors in the precipitation of coronary thrombi. Such a conclusion implies that coronary thrombi

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immediately prior to the heading "Summary and Conclusions," said :—"What I think the writer means is that capillaries may give way in or near the atheromatous patch which tend then to cause some projection into the lumen. That is similar to the idea of Winton. He thinks that stress or strain might aid such haemorrhages. That is physical or emotional strain or a high pressure. You get then a disposition of platelets from the blood stream which go on accumulating over a period of several days and then you may get a sudden clot formed more particularly of red cells which may lead to blockage and with that blockage you get the sudden pain or collapse or whatever it may be. That is why he says that it is not what the individual has been doing immediately prior to the catastrophe but what he has been doing during the last two or three days which are contributory factors if he is correct in assisting the strain. I think as far as I can see the evidence of the times in which these cases occur does not seem to associate the onset with effort. The statistics I have presented show the time when severe symptoms first developed in a series of patients in whom the disease was not immediately mortal. Obviously at the Adelaide Hospital it is only an accident when a patient dies in the hospital immediately of coronary disease. The impression is and we mentioned it to students that coronary thrombosis does not seem to be related to effort.

are initiated and progress to the point of occlusion in a short space of time—an inference that has no pathologic basis. Actually there is definite evidence to prove that hours or even days elapse between the time of the inception of the thrombus and the moment when occlusion, with its resulting cardiac pain, occurs. To demonstrate this, one has only to study the structure of the occluding thrombus in a person who, previously in apparently good health, collapses and dies with the onset of cardiac pain and before infarction has had time to take place If a serial section is made through the entire length of the thrombus, some levels will show the process to be many hours or days old. The following two cases illustrate the gradual nature of the formation of a thrombus: Case 1. A man aged fifty-four collapsed suddenly on the street and died before he could be hospitalized. No previous history was obtained. Autopsy revealed marked coronary sclerosis and recent thrombosis of the left anterior descending coronary branch. The left circumflex artery, at its immediate origin, showed a localized area of intimal haemorrhage without thrombosis of the adjacent lumen. The thrombus in the left anterior descending branch measured 1.5 cm. in length. The affected part of this artery was embedded in one block and sectioned serially at intervals of 7 microns from one end to the other, the sections being cut horizontally. Every section was stained and examined. At certain levels the thrombus consisted, morphologically, of two distinct portions. First there was a small, oval, radiating mass of condensed material which was localized to one side of the lumen and which showed invasion by fibroblasts in some sections. Second there were masses of more loosely arranged networks of fibrin and platelets in which red cells and leukocytes were enmeshed. In many of the sections only the latter type of thrombus material could be found and in some it occupied the entire lumen. The older portion of the thrombus was attached to the endothelium immediately adjacent to a point of marked haemorrhage into the inner aspect of an atheromatous focus. The serial sections failed to reveal a break in the tissues lying between the

. . . Coronary thrombosis does not necessarily cause death. . . . Although in a post-mortem a clot is found the clot does not always cause death. They may get pneumonia or something or in some other way. From a sudden strain they may have a sudden spasm from the artery beyond. It is hard to disentangle the heart failure directly due to the thrombosis from a superadded spasm of the vessel. . . . If a man is found dead and a clot is found and there is no other apparent reason for the death it would be attributed to the clot. . . . I think the clot in Forst's case was formed through capillary haemorrhages in a pre-existent atheromatous patch (that would be why the haemorrhages would occur) and this had caused some raising of the patch possibly rupture, rupture and deposition of platelets and then finally the clot in the lumen. The capillaries may come either from the lumen or near the lumen or rather from the orifices of branches of the vessel, or from the adventitia. They may come either from the lumen side or outside the coronary vessels. *Winton* stresses that when a branch occurs at the coronary artery . . . the capillary may also pass from the mouth of that branch into the intima. I presume that the pressure within the capillaries would vary with the pressure in the lumen but that is more for the physiologist. The occlusion or partial occlusion of the lumen by the clot would affect the supply of blood to the heart,

haemorrhage and the thrombus, nor was there any atheromatous material incorporated in the thrombus. At the point of attachment of the older thrombus mass a rounded structure resembling a thrombosed capillary was seen in the subendothelial tissue. Otherwise there was little evidence of intimal vascularization. The occluding thrombus lay on one side of a point of stenosis of the lumen, but the relation of the stenosing plaque to the direction of blood flow was not ascertained. Case 2. A man aged sixty-eight was found dead in a hotel bedroom. He was fully dressed and was kneeling at the side of his bed. From the degree of rigor mortis that had set in it was estimated that death had occurred about three and one-half hours after he had left work, at which time he had not complained of any pain but appeared to be in his usual state of good health. Autopsy revealed a recent thrombus occluding the left circumflex coronary artery at a point about 0.7 cm. from its origin. Immediately proximal to the thrombus the lumen was markedly stenosed by an atheromatous plaque. At the distal extremity of the thrombus the intima was slightly raised by a small, reddish black mass which appeared to be a haemorrhage into an atheromatous focus. A similar but larger intimal haemorrhage was noted in the left anterior descending branch about 1 cm. from its origin. The endothelium overlying both intimal haemorrhages appeared to be intact. The haemorrhage in the left anterior descending branch showed no gross evidence of thrombus formation in association with it nor was there any stenosis of the lumen in its vicinity. The thrombosed portion of the left circumflex artery was embedded in one block and sectioned serially at intervals of 10 microns throughout its entire extent. The sections were cut longitudinally in an attempt to demonstrate the relation of the occluding thrombus to the point of stenosis previously mentioned. Sections were mounted and stained at intervals of about 200 microns. Microscopically the thrombus consisted, first, of an older organizing portion which was adherent to the intima immediately distal to the apex of the stenosing atheromatous plaque. From the amount of fibroblastic prolifer-

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either cutting off the blood supply leading to the muscle being unable to work or to lessening the reserve power of the heart. Supposing there is just a partial occlusion the individual might get on all right, . . . but some unusual effort might be too much for the heart. In Forst's case I think the final clot formed very quickly but the antecedent changes might have occupied several days. That is the platelets forming on the injured surface. The gradual silting up of portion of the vessel depends on the size of the vessel. People with large coronary vessels and those who have hyper-pressure hearts would have correspondingly large coronary vessels and would be less affected by a small clot in portion of the wall than a person with a small coronary artery where the corresponding change would reduce the amount of blood passing through relatively. This clot was partially occluding the coronary vessel. The antecedent changes would protrude into the lumen and if sufficiently large would affect the blood supply to the heart. If you had a sufficient projection you will reach a stage when the reserve power of that artery may not stand up so well to sudden strain. In Forst's case he could not have done any severe exercise with that portion of the vessel nearly occluded or one would not expect him to do much severe exercise and that is why I think that the final clot that killed him was formed rapidly immediately prior to his

ation in this part of the thrombus there is reason to believe that it was at least three days old. Secondly there was a mass of fibrin and platelets with enmeshed red cells which was attached to the older thrombus and which apparently completed the occluding process. Two points of microscopic haemorrhage were noted in the intima underlying the thrombus. Immediately below the attachment of the older portion of the thrombus there was a mass of pink-staining organizing material which resembled clotted blood and in which a large amount of hemosiderin was demonstrated by Perl's stain. A few small capillaries were seen in this area of old haemorrhage. Distal to the entire thrombus mass, and apparently in no relation to it, the endothelium was elevated by an intimal haemorrhage in which the red cells were intact. Random sections made through the discrete haemorrhage noted in the left anterior descending branch showed the adjacent lumen to be free from thrombus formation. This haemorrhage had occurred into a large atheromatous focus, and in the sections studied it was separated from the lumen by a thin layer of dense tissue. In the latter layer, numerous capillaries were seen to enter the haemorrhage focus and to run in the direction of the lumen, although their actual connection with the lumen was not demonstrated. Because of the evidence of organization, i.e., invasion by fibroblasts, in the older portion of the thrombus in each of these two cases the inception of a thrombus must have occurred at least three days before death. There is no reason to believe that any unusual symptoms were experienced either at the time of the initial thrombus inception or in the ensuing latent period. Both persons were pursuing their ordinary activities until just before their fatal attacks, and patient 2 was known to have been without complaints and in good spirits three and one-half hours before his death. In this case microscopic examination of that part of the myocardium supplied by the occluded artery failed to reveal any evidence of infarction. It may be concluded, therefore, that death had followed coronary occlusion so rapidly that sufficient time had not elapsed for infarction to occur. In other words, this case does not belong to the

death. A good deal depends on whether the other parts of the coronary artery are healthy. The death would follow the formation of the clot within a minute or so. I remember the exertion that Forst had been engaged in. It is my opinion that the final clot formed after he came down from the crane. I think that as soon as the clot formed he would be unable to do anything. I can't see any special connection between the work done and the formation of the clot. At first sight one would naturally be inclined to associate any unusual effort with the fatal result. That is I would. I would try and see some reasonable association between the two but in the case of thrombosis in the coronary artery one's previous experiences being taken into consideration one fails to see an association that appeals to one as being right. If you hold your breath I understand that your pulse becomes weak. I understand that it would be expected to slow the circulation if the exercise done by Forst involved his holding his breath. That would facilitate clot formation. If the clot had formed on the jib he would have fallen down. The slowing of the circulation tends to let the platelets drop out and it would facilitate the formation of the clot. Whether the hanging on to the rope would affect this would depend on whether or not he held his breath. Referring to list of statistics. In the list of cases the patients' first onset was at 11 p.m. and the patient died eight

'silent' group in which coronary thrombosis with infarction occurs without definite signs and symptoms resulting. The immediate cause of death in each of the two cases appeared to be the terminal occlusion of the coronary lumen by a recently formed mass of thrombus material superimposed on and propagated from an older thrombus which was at least three days old. The formation of a thrombus in these two persons, then, was a gradual and not a rapid process. Therefore, to eliminate physical exertion or emotion as a precipitating factor in coronary thrombosis, the activities of the patient should be investigated not only for the few hours prior to the attack but for many days previously. On purely pathologic grounds there is reason to believe that such physical and mental states, both of which result in temporary hypertension, cannot be excluded as precipitating factors of coronary thrombi. In both of the cases reported here, as well as in thirty-one of thirty-six consecutive cases reported elsewhere, haemorrhagic foci were seen in the intima at the site of thrombotic occlusion. The mechanism of production of intimal haemorrhage has already been described in detail, my original observations and in part my conclusions having been confirmed by *Wartman* and by *Winternitz* and his co-workers. Stated briefly, this lesion results not from the backflow of blood through an intimal defect produced by the rupture of an atheromatous 'abscess,' as was previously thought, but from the rupture of discrete capillaries which are derived from the coronary lumen. Intimal capillaries are a common finding in the tissues adjoining most intimal haemorrhages, and if a careful section is done they may sometimes be shown to arise from the lumen of the artery and to enter the haemorrhagic focus. Capillary rupture with ensuing haemorrhage into the intima is not confined always to the points of thrombotic occlusion. The haemorrhagic lesions are frequently multiple, as in the two cases cited in this report, and they may and do occur without associated thrombosis of the adjacent coronary lumen. However, it has been suggested that if the haemorrhage with its concomitant tissue damage occurs at a point at which a

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days later when sitting on a bed-pan. I don't know anything more of the history of that case. He would not be likely to be constipated as he would probably have had purgatives. The straining on the bed-pan would produce the same sort of result as holding the breath. That straining might be hard work. Cases have occurred where people have collapsed. It may be the sitting up in bed just as much as the effort. I have read the whole of the article of *Paterson*. I agree with the following sentences from this article :—' Therefore it seems logical to assume that intimal capillaries because of their peculiar position will be sensitive to sudden increases in the coronary blood pressure. If such an increase in the coronary blood pressure occurs these capillaries will be in imminent danger of rupture particularly if the surrounding tissues are in a state of laxity from atheromatous degeneration. Sudden and temporary increases in the coronary blood pressure are encountered commonly in circumstances of unusual exertion and emotion.' Later on in the article appears the passage I have referred to. I understand that coronary thrombosis is generally evidenced by certain symptoms but silent infarctions may occur. You do get patients coming to post-mortems who have had no severe illness at all and therefore with only trifling symptoms as indigestion. If time permitted you would expect severe pain but in these cases of sudden death . . . sometimes there is not time to express pain and sometimes the patient is *in articulo*

stenosing arteriosclerotic plaque has produced stagnation and eddying of blood, conditions may then be favourable for the deposition of a thrombus. In the final analysis, then, the factors responsible for the rupture of intimal capillaries should be the immediate causes of the precipitation of coronary thrombi. Two principal factors appear to be involved in the mechanism of capillary rupture : (1) softening, by atheroma, of the tissues surrounding and supporting the capillary wall, and (2) high intracapillary blood-pressure. The influence of atheromatous degeneration on the production of capillary rupture has been described in detail elsewhere. Intimal haemorrhages have been found to occur almost exclusively in areas of atheromatous degeneration. It is assumed that softening, which is a physical character of atheroma, allows the pressure of blood within the capillary to dilate its walls to such an extent that rupture eventually occurs. This assumption is borne out by the fact that intimal capillaries are usually of small calibre as they traverse the denser intimal layers, while they are frequently dilated in areas of atheroma. Furthermore, the age incidence of coronary thrombosis and intimal haemorrhage corresponds roughly with that in which atheroma usually develops, i.e., late middle age. Younger persons with characteristically dense and fibrous arteriosclerotic lesions, and elderly persons with heavily calcified plaques, are not so prone either to intimal haemorrhage or to coronary thrombosis. The factor of high intracapillary blood pressure as a cause of capillary rupture is equally important, and it is with this factor that excessive exercise or emotion is concerned. Intimal capillaries, because they arise directly from the main coronary lumen, are exposed constantly to a relatively high pressure of blood. They are not, like other capillaries, at the end of a long series of arteries, and arterioles which absorb much of the pressure by friction. Therefore it seems logical to assume that intimal capillaries, because of their peculiar position, will be sensitive to sudden increases in the coronary blood pressure. If such an increase in the coronary blood-pressure occurs, these capillaries

mortis and unable to do so. Assuming in this case that there were no symptoms at all, I would not like to express an opinion as to whether death was due to coronary thrombosis."

The arbitrator found that Forst's death was due to coronary thrombosis, but he was of opinion that the widow had failed to prove that the death was due to his exertion above described or to the work which he was doing; and he found that the workman's death was not caused by injury by accident arising out of and in the course of his employment.

Under sec. 41 of the Act an appeal lay to the Supreme Court as well upon matters of fact as upon matters of law. The widow appealed, and the Full Court allowed her appeal, set aside the arbitrator's award, found that the workman's death was the result of injury by accident arising out of and in the course of his employment, and remitted the application for compensation to the arbitrator.

Material passages from the reasons for judgment of the arbitrator and of the Supreme Court are cited in the judgments hereunder, and, in particular, in the judgment of *McTiernan* J.

The company appealed to the High Court.

Ligertwood K.C. (with him *V. R. Millhouse*), for the appellant. The Full Court, relying on a passage in Professor Cleland's evidence, drew an inference, and based its judgment on an inference, which Professor Cleland, an expert witness, himself refused to draw. This

will be in imminent danger of dilatation and rupture, particularly if the surrounding tissues are in a state of laxity from atheromatous degeneration. Sudden and temporary increases in the coronary blood-pressure are encountered commonly in circumstances of unusual exertion and emotion. With strenuous muscular exercise the systolic blood-pressure is said to rise to from 160 to 180 mm. of mercury. A corresponding rise in the diastolic pressure, although to a lesser degree, also occurs. Emotional stress, often but not always, results in a similar sudden increase in the systolic and diastolic pressures. For example, I have heard of a marked increase in the blood-pressure of a young woman each time she recounted the details of a particularly harrowing experience. This woman showed a normal variation in blood-pressure of from 120 to 140 mm. systolic and from 80 to 95 mm. diastolic. During an emotional upset the systolic pressure varied from 150 to 180 mm. and the diastolic from 100 to 120 mm. It is suggested, therefore, that high coronary blood-pressure, the result of strenuous exercise or of emotion (or of persistent hypertension), is one of the underlying causes of capillary rupture and intimal haemorrhage in arteriosclerotic coronary arteries. If conditions are favourable, the various changes which result from capillary rupture may then be the initiating factors in the disposition of coronary thrombi.

SUMMARY AND CONCLUSIONS.

The formation of coronary thrombi is a gradual process, sometimes occupying several days before occlusion of the coronary lumen with its resulting cardiac pain is produced. Therefore the activities of a patient immediately preceding the onset of an attack of coronary thrombosis have no relation to the aetiology of the precipitation of a thrombus but are purely coincidental. The pathologic appearances in a series of fatal cases of coronary thrombosis suggests strongly that excessive exercise and emotional stress are intimately concerned in the mechanism of coronary artery thrombosis."

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inference was not accepted by the expert. The arbitrator's reasons for judgment contained a very strong finding on the facts before him, and such a finding should not be upset by this court. Rival theories of expert witnesses were properly regarded as conflicting evidence of fact, and the arbitrator's findings should be treated as any other findings of fact. [He referred to *Hetherington* v. *Amalgamated Collieries of W.A. Ltd.* (1); *Gwyer* v. *Auckland Harbor Board* (2); *Wynyard* v. *Daily Telegraph Co. Ltd.* (3); *Jarvis* v. *One Tree Hill Borough* (4); *Powell* v. *Streatham Manor Nursing Home* (5); Dr. *J. C. Paterson's* article in the *Journal of the American Medical Association* (March 1939).*]

Cleland K.C. (with him *Teesdale Smith*), for the respondent. Dr. *Paterson's* article was not published until near the end of the arbitration, and it was apparently considered by no witness except Professor Cleland. Had the magistrate understood and applied this article, his opinion would have been different. The medical evidence does not show that coronary thrombosis is never caused by exertion. The whole of the facts immediately antecedent to the death should be considered, and on the balance of probabilities it should be said that the workman's exertion contributed in a material degree to the conditions from which his death arose. In particular, the final clot appears to have formed very quickly, and what is more probable than that it was caused by the strain which Forst incurred? [He referred to *Falmouth Docks and Engineering Co. Ltd.* v. *Treloar* (6).]

Ligertwood K.C., in reply.

Cur. adv. vult.

Dec. 19.

The following written judgments were delivered:—

RICH A.C.J. This is an appeal from a decision of the Supreme Court of South Australia consisting of *Angas Parsons* and *Napier* JJ., who allowed an appeal from an award of a special magistrate sitting under the *Workmen's Compensation Act* 1932-1935 (S.A.).

The special magistrate's award refused compensation to the now respondent, the widow of a deceased workman who collapsed while at work and died. A post-mortem examination disclosed evidence of a coronary thrombosis which would cause occlusion and consequent cardiac insufficiency. Having heard an abundance of medical

* See, *ante*, footnote to pp. 555 et seq.

(1) (1939) 62 C.L.R. 317.
(2) (1937) N.Z.L.R. 808.
(3) (1934) N.Z.L.R. Supp. 137.
(4) (1940) N.Z.L.R. 280.
(5) (1935) A.C. 243, at p. 249.
(6) (1933) A.C. 481.

evidence from witnesses whose attainments and eminence neither were nor could be challenged, but whose opinions exhibited no greater degree of unanimity than is commonly met with in other departments of abstruse knowledge and of scientific research, the learned magistrate arrived at a negative conclusion. His finding was, in substance, that coronary thrombosis could not, in the present state of knowledge, be connected with exertion and accordingly that it was not established that the workman's employment materially contributed to the cause of his death.

The learned judges of the Full Court considered the whole of the medical evidence, as, under the Act, they are entitled to do, and, having described the duty of the court to arrive at some conclusion on an issue of fact, however "difficult or invidious" it might be made by the state of scientific knowledge and opinion, their Honours proceeded, by a course of reasoning which combined common sense with the application of logic to physiological facts, to infer "on the preponderance of probabilities" that the thrombus was precipitated as the result, in part, of some unusual exertion undertaken by the workman before his collapse.

In my opinion the conclusion of the Full Court is correct. I am greatly impressed by the sequence of events. The deceased, who had arrived at an age when arterio-sclerosis and atheroma afflict mankind, was a stevedore's labourer. On the day of his death he climbed up the jib of the crane and lay prone on the crane with his arms outstretched, trying to replace a wire which had come off the gin. He failed to do so, returned to the deck and for some time, with his arms in a position raised over his head, helped in holding up a wire rope. Immediately after performing this task he collapsed. What weighs so much with me is the fact that he was brought to a standstill, as an ordinary lay observer would think, by the exertion he had undergone: Cf. *Partridge Jones and John Paton Ltd.* v. *James* (1). I do not see why a court should not begin its investigation, i.e., before hearing any medical testimony, from the standpoint of the presumptive inference which this sequence of events would naturally inspire in the mind of any common-sense person uninstructed in pathology. When he finds that a workman of the not-so-young standing attempts in a posture calculated by reason of the pressure on the stomach to disturb or arrest the rhythm of the heart a very strenuous task not forming part of his ordinary work and then collapses almost immediately and dies from a heart condition, why should not a court say that here is strong ground for a preliminary presumption of fact in favour of the view

(1) (1933) A.C. 501, at p. 505.

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that the work materially contributed to the cause of death? From this standpoint the investigation of physiological and pathological opinion shows no more than the current medical views find insufficient reason for connecting coronary thrombosis with effort. Be it so. That to my mind is not enough to overturn or rebut the presumption which flows from the observed sequence of events. If medical knowledge develops strong positive reasons for saying that the lay common-sense presumption is wrong, the courts, no doubt, would gladly give effect to this affirmative information. But, while science presents us with no more than a blank negation, we can only await its positive results and in the meantime act on our own intuitive inferences. The conclusion of the special magistrate may prove to be in advance of its time, but, as matters stand, I prefer that of the Full Court.

In my opinion the appeal should be dismissed.

STARKE J. Appeal from a judgment of the Supreme Court of South Australia, which declared that the appellant was liable to pay compensation under the *Workmen's Compensation Act* 1932-1935 to the respondent, the widow of Carl H. M. Forst and sole dependant of Forst, in respect of an injury by accident arising out of and in the course of his employment, and discharging and setting aside an award to the contrary in an arbitration under the Act.

Forst was employed by the appellant as a waterside worker. He was engaged in June 1938 in discharging cargo from the s.s. *Maloja*. He started work at 8 a.m., and throughout the day, until he died at about 4 p.m., he appeared to be in normal health. At about 4 p.m. the wire of the crane that he was working ran off its sheave, and when that happened Forst climbed up the lattice-work of the crane and attempted to replace the wire. It was a job which required muscular exertion, and he was in an awkward position to perform it. He was unsuccessful and decided to leave the job to the crew, to whom it strictly belonged. So he came down. He was then ordered to assist a fellow workman to pull down the wire of another crane. This work also needed effort. He had to pull the wire with his hands over his head, pulling down each hand alternately until his hand would be somewhere near the level of his face. Forst walked away when the pulling was for all practical purposes complete, and within a few seconds, during which time he walked about twelve yards, he fell down, lost consciousness, and died within a very short time.

The arbitrator found, and the Supreme Court affirmed his finding, that Forst's death was due to coronary thrombosis. There is ample

evidence to support this finding and no reason that would justify this court in overruling the concurrent finding of fact of the arbitrator and the Supreme Court.

It is now well settled that, when a man in a diseased condition dies, and it is found that the disease and the work contribute to his death, then his death results from accident within the meaning of the Act (*Partridge Jones and John Paton Ltd.* v. *James* (1); *Hetherington* v. *Amalgamated Collieries of W.A. Ltd.* (2)). The question is in all cases one of fact: Cf. *Jones* v. *Blaenavon Co. Ltd.* (3).

The arbitrator, however, found that "on the science of medicine . . . death from coronary thrombosis cannot generally be related to exertion but that death in such cases is a mere happening in the chronological order of things. If death does occur concurrently with or closely connected in point of time with exertion by Forst, I am unable . . . to connect the two in such a way as to say that exertion caused the death. . . . In other words, merely because shortly before his death Forst was exerting himself and shortly afterwards died of coronary thrombosis does not cause me to reach the conclusion that the exertion had anything to do with his death."

The Supreme Court, to which an appeal lay as well upon matters of fact as upon matters of law (Act, sec. 41), reached a different conclusion. "We agree," said the learned judges, "that . . . it must be difficult and probably impossible to relate an attack of coronary thrombosis to any particular activity of the patient and we concede that even in this case it is difficult to feel any great confidence in the inference but it seems to us that we have here the coincidence of strenuous physical exertion occurring at or about the time when the thrombosis must have commenced to precipitate. If the expert evidence shows, as we think it does, that physical effort is commonly—although not invariably—the inciting cause of that phenomenon, we think that we are entitled to draw the inference that" a medical witness "felt unable to draw. We fully accept his view that the evidence is in some measure inconclusive. For the purpose of a scientific deduction it may be insufficient, but we repeat that courts of justice are entitled and bound to act upon the probabilities of the case."

It is thus apparent that the determination of the question of fact in this case is not easy. Medical science gives no certain answer to the question, and the medical witnesses differ among themselves as to the proper conclusion in this case. The medical aspects of the class

(1) (1933) A.C. 501. (2) (1939) 62 C.L.R. 317.
(3) (1931) 24 B.W.C.C. 148.

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of cases now under consideration have, I think, had more detailed consideration in New Zealand than elsewhere, as is suggested by the following cases: *Harvey* v. *E. & H. Craig Ltd.* (1); *Wynyard* v. *Daily Telegraph Co. Ltd.* (2); *Gwyer* v. *Auckland Harbour Board* (3); *Jarvis* v. *One Tree Hill Borough* (4). "Until quite recently it was generally agreed that excessive physical exertion or emotion was intimately connected with the precipitation of coronary thrombi." This statement is taken from an article by Dr. *J. C. Paterson* in the *Journal of the American Medical Association* of March 1939: See also the report of Dr. *Fitchett* (5). And two of the medical witnesses called in the present case still, I think, adhere to it, namely, Drs. Cherry and Covernton. Medical opinion recognizes that the precipitation of coronary thrombi is not always due to effort or exertion. And many eminent men, I gather, are of opinion that effort and exertion does not contribute to and is not connected with such precipitation. Dr. Gartrell expounded this view. On the other hand, Dr. Paterson in his paper suggests that high coronary blood pressure, the result of strenuous exercise or of emotion (or of persistent hypertension), is one of the underlying causes of capillary rupture and intimal haemorrhage in arteriosclerotic coronary arteries. If conditions are favourable, he suggests that the various changes which result from capillary rupture may then be the initiating factors in the disposition of coronary thrombi. He thus summarizes his views and conclusions:—"The formation of coronary thrombi is a gradual process, sometimes occupying several days before occlusion of the coronary lumen, with its resultant cardiac pain, is produced. Therefore the activities of a patient immediately preceding the onset of an attack of coronary thrombosis have no relation to the etiology of the precipitation of a thrombus but are purely coincidental. The pathologic appearance in a series of fatal cases of coronary thrombosis suggests strongly that excessive exercise and emotional stress are intimately concerned in the mechanism of coronary artery thrombosis." Both Professors Cleland and Hicks of the Adelaide University were impressed, I think, with this theory. Another theory may be found in the report of Dr. *Fitchett* (6), that effort or exertion provoke a demand from the heart for extra blood, which defective coronary arteries cannot supply, thus inducing a relative ischaemia and exciting ventricular fibrillation which leads in a material degree to a fatal issue. But, in all this uncertainty, there is a passage

(1) (1933) N.Z.L.R. Supp. 102.
(2) (1934) N.Z.L.R. Supp. 137.
(3) (1937) N.Z.L.R. 808.
(4) (1940) N.Z.L.R. 280.
(5) (1934) N.Z.L.R. Supp., at p. 147.
(6) (1934) N.Z.L.R. Supp., at pp. 138-150.

in Professor Hicks' evidence which appeals to my mind and justifies the conclusion that Forst's death was due to an accident, in the relevant sense, which arose out of and in the course of his employment. "In my opinion," he said, "assuming that it was ante-mortem clot I think it is ignoring too great a functional disturbance occasioned by this effort to say that the formation of the clot was not due to disturbances produced by that effort. The effort in other words is the most noteworthy fact in relation to this cardiac death on the day of the death."

In the present case the deceased was engaged upon heavy work and, whilst so engaged, collapsed and died almost immediately. A post-mortem examination disclosed a thrombus three-quarters of an inch from the orifice of the coronary artery descending along the front of the heart. The clot not only occupied part of the lumen of the artery but extended through an aperture in the inner lining of the wall, being continuous with a further clot situated between the layers of the wall of the coronary artery and underneath a patch of that wall, which was affected by atheroma. The clot must have been quite recent, said Dr. Gartrell, and formed within hours of death and certainly before death, because, unless there had been this aperture leading from the lumen of the artery underneath the diseased patch, there would not be any blood underneath that layer to form a clot *post mortem*. All this satisfies me, as it satisfied the Supreme Court, that the functional disturbance occasioned by the work of the deceased was intimately connected with and contributed to the conditions observed upon the post-mortem examination.

The appeal should be dismissed.

Dixon J. By the order under appeal the Supreme Court of South Australia allowed an appeal from a special magistrate presiding at a Local Court and set aside his decision or award refusing a claim under the *Workmen's Compensation Act* 1932-1935 and in lieu thereof declared that the employer (who is the appellant in this court) was liable to pay compensation under that Act to a deceased workman's widow, the respondent.

The workman died, as it has been found, in consequence of a coronary thrombosis. The finding that the cause was a thrombosis has been sustained by the Supreme Court.

Immediately before his collapse the deceased workman had undergone some exertion unusual in kind and degree. The question now at issue is whether the special magistrate ought to have found that the work done by the deceased, and, in particular, the exertion, contributed in any material degree to the condition from which his

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death so arose. The Supreme Court held that such a finding should be made.

If the work done by the deceased did contribute in any material degree to the formation of the thrombosis, then, as the law has been interpreted, the facts would warrant the conclusion that he died by accident arising out of and in the course of his employment. In *Hetherington* v. *Amalgamated Collieries of W.A. Ltd.* (1) the court examined the state of the law governing a claim that death from coronary occlusion or cardiac insufficiency amounts to death by accident arising out of and in the course of employment. The issue of fact is defined by that decision and by the cases upon which it depends, and it is unnecessary now to say more than that the issue involves a medical question which must be decided as a matter of fact upon the evidence adduced in each given case. The issue having been defined, it is impossible to treat the question raised as anything but an unmixed question of fact, medical and scientific in character, and therefore to be decided upon expert testimony. No doubt such cases fall into a well-defined class and raise the same problem or similar problems, and it may be a matter for regret that the courts are unable to establish an absolute rule by which the decision of every such case would be governed. But the problems are medical, and the fact that in the present state of medical knowledge and opinion a uniform and decisive answer cannot be given on each occasion when the question is raised is anything but a ground for the courts of law attempting to supply by legal reasoning a solution to what is entirely a question of fact. The difficulty can be met only by legislation.

In the present case the special magistrate heard and considered testimony ably given by experts of great knowledge and experience. How far work or exertion brings about or contributes to coronary thrombosis was one of the matters to which that testimony was directed. The learned special magistrate's conclusion was "that death in such cases is a mere happening in the chronological order of things. If death does occur concurrently with or closely connected in point of time with exertion, it cannot satisfactorily be said to have been caused by such exertion." This conclusion appears to me to be fully supported by the evidence and to represent what, according to the evidence, would seem to be the fair result of competent medical opinion at the present time.

I think the special magistrate's finding gives proper effect to the evidence adduced. In the Supreme Court, *Angas Parsons* and

(1) (1939) 62 C.L.R. 317.

Napier JJ., after a close consideration of the evidence, formed the contrary view. Their Honours based their opinion in no small degree upon deductions, which they drew, of a physiological and pathological nature. Professor Cleland, the Professor of Pathology in the University of Adelaide, had said in evidence that he thought, as far as he could see, that the evidence of the times in which such cases occurred did not seem to associate the onset with effort and that the impression was (and it was mentioned to students) that coronary thrombosis does not seem to be related to effort. In reference to this statement, their Honours say : " It may be difficult for us to draw an inference that medical men hesitate to draw, but we do not think that we can refuse to enter upon an inquiry because it is difficult or invidious, and it seems to us that Professor Cleland has given us the material for a conclusion." The judgment then sets out a course of reasoning of a medical nature, some of the materials for which are drawn from contributions to medical literature that were in evidence and contained an exposition which Professor Cleland appeared to their Honours to accept. After citing a passage from Professor Cleland's evidence, the judgment continues as follows :—" We agree with the witness that, speaking generally, it must be difficult and probably impossible to relate an attack of coronary thrombosis to any particular activity of the patient, and we concede that, even in this case, it is difficult to feel any great confidence in the inference, but it seems to us that we have here the coincidence of strenuous physical exertion occurring at or about the time when the thrombus must have commenced to precipitate. If the expert evidence shows, as we think it does, that physical effort is commonly—though not invariably—the inciting cause of that phenomenon, we think we are entitled to draw the inference that Professor Cleland felt unable to draw. We fully accept his view that the evidence is in some measure inconclusive. For the purposes of a scientific deduction it may be insufficient, but we repeat that courts of justice are entitled and bound to act upon the probabilities of the case." The difficulty I feel in adopting these views lies in three considerations.

First, I think that upon a question of fact of a medical or scientific description a court can only say that the burden of proof has not been discharged where, upon the evidence, it appears that the present state of knowledge does not admit of an affirmative answer and that competent and trustworthy expert opinion regards an affirmative answer as lacking justification, either as a probable inference or as an accepted hypothesis.

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Secondly, I do not read the evidence, considered as a whole, as meaning that physical effort is commonly, although not invariably, the inciting cause of coronary thrombosis.

Thirdly, whether an inference can or should be drawn from the fact that in the present case the collapse of the deceased occurred after unusual exertion seems to me to depend on the answer first given to the pathological question whether there is any natural connection between exertion and the formation of a thrombus.

Tempting as it always is, particularly in matters of bodily health, to argue from a sequence of external events, such reasoning is justified only when positive knowledge or common experience supplies some adequate ground for believing that the events are naturally associated. The evidence upon which the special magistrate acted is to the effect that there is no such ground.

For these reasons I think that the appeal should be allowed.

McTIERNAN J. The question in this case is whether the Supreme Court was in error in reversing the decision of the court of first instance, which found that the death of the respondent's husband did not result from personal injury by accident arising out of and in the course of his employment with the appellant. The decision of the Supreme Court was given upon an appeal which it entertained under sec. 41 of the *Workmen's Compensation Act* 1932-1935 of South Australia. This section gives the Supreme Court jurisdiction to decide questions of fact and law and to rehear the case.

There are concurrent findings of the two courts that the cause of death was coronary thrombosis. The widow's advisers put forward that death was due to another cause, which is more plainly connected with physical exertion. There is ample evidence to support the conclusion at which both courts have arrived as to the cause of death, and I do not think that it ought to be disturbed.

Coronary thrombosis comes within the definition of "personal injury by accident arising out of and in the course of the employment" (*Hetherington* v. *Amalgamated Collieries of W.A. Ltd.* (1))—See also *Falmouth Docks and Engineering Co. Ltd.* v. *Treloar* (2).

The court of first instance and the Supreme Court reached opposite conclusions on the question whether the deceased's work accelerated the progress of the disease, which the post-mortem examination revealed was affecting his heart, to the condition resulting in his death.

We have now to decide whether the evidence justifies the inference that the exertion brought about by the strenuous work done by the

(1) (1939) 62 C.L.R. 317. (2) (1933) A.C., at p. 486.

deceased immediately before he died accelerated or materially contributed to his death. The appellant contends that the inference is not justified because it is equally probable or more probable that the artery leading to the heart became occluded without any extraneous causation but merely by the progress of the disease from which the deceased was suffering.

It was proved that he was apparently a strong energetic man, and on the day on which he died he had worked with his normal vigour and efficiency as a winchman in the appellant's employment. He had worked on that day from 8 a.m. until 4.15 p.m., when he collapsed and died. He had not during the day exhibited any sign of pain or distress.

The special magistrate who constituted the court of first instance described the particular operations which the deceased carried out immediately before he died in these words :—" Forst was a winchman —a powerful man weighing fifteen to sixteen stone. On the 4th June 1938 he was on the *Maloja*. About four o'clock in the afternoon the wire ran off one of the sheaves on the crane which Forst was operating. He stopped operations and climbed up the crane to lift the wire on the sheaf. The crane was then at an angle of about forty-five degrees. Forst lay along the crane and taking hold of the wire tried to lift it and shove it at the same time to lift it over the gin. After two or three unsuccessful attempts he came down again and gave every appearance of being quite normal. He came down because he found it was going to be difficult, and he would leave the job to the crew whose job strictly it was. It was a job which required muscular exertion and the man was in an awkward position to perform it. He then spoke about the quantity of cargo to Harmsdorf who had worked with him at different times for years. The foreman Hodgson then instructed him to assist Strudwick to pull down the wire of another crane. He then wheeled around quite normally, and began that job which meant that he had to pull the wire with his hands over his head pulling down each hand alternately until his hand would be somewhere near the level of his face. This work again needed effort. It was not easy. Strudwick had been on it and Forst had been sent to lighten the latter's task, and finally a Lascar joined in. . . . With the pulling for all practical purposes completed Forst walked away. Strudwick who was working face to face with him noticed nothing wrong with him. Within a few seconds of his ceasing work during which time he walked about twelve yards he fell down. He hit and cut his head in falling but there is no evidence of fracture of

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the skull. Apparently Forst did not regain consciousness and it is clear on the evidence that his death was due to cessation of heart action." There would be no doubt that all these facts would amply justify the inference that the work that the deceased did immediately before he collapsed accelerated or materially contributed to his death if there was not a strong body of medical evidence that physical exertion is not required to cause the fatal onset of coronary thrombosis.

The weight to be given to the facts of this case standing alone is well illustrated by the observations of Lords *Loreburn* and *Atkinson* respectively in *Woods* v. *Thomas Wilson Sons and Co. Ltd.* (1). It is sufficient to quote the views of Lord *Loreburn* :—" He " (the County-Court judge) " had to choose between a variety of possible explanations, and he found that ' the physical condition of the man at his age would be likely to produce, and did produce, a weakened bowel, and that an accident of this sort is just the sort of thing to have caused, and did cause, acute injury to the weakened bowel which otherwise might have lasted for a considerable time and not interfered with his efficient work, and that the injury so caused gradually produced perforation and so accelerated his death.' In choosing between the view that this blow accelerated death and the view that it did not, he looked at events as well as opinions : ' The evidence appears to me to be overwhelming that before the accident he was an efficient workman though old, and that immediately after it he was completely ineffective and thrown into such a condition that he first collapsed and then died within a few days. I come to the conclusion, therefore, that his death was accelerated, that is to say caused, by the injury resulting from the accident.' In my opinion that ends this case. To my mind this reasoning was quite legitimate. The blow was instantly followed by disablement from work and by pain in the region struck and almost immediately by vomiting. Pain in the same region and vomiting continued to some extent at all events, and in a week the man died. If there were nothing else, I should say that these facts were evidence of an injury by accident and a

(1) (1915) 8 B.W.C.C. 288, at pp. 295, 296, 300.

resulting death. It is conceivable, as counsel argued, that the commencement of the illness, though simultaneous with the blow, may have been due simply to appendicitis seizing him at that precise moment. It would be a curious coincidence. It seems infinitely more probable that the commencement of the illness was due to the blow. But there is something else, namely, the medical evidence both of physical fact and of opinion. The doctors somewhat differ as to the inference they draw from those physical facts, but two of them think that the condition which led to death might have been brought about by a blow. This does not displace or contradict, but in some degree confirms, the conclusion which would be warranted from the story of what happened to the man and how it affected him on the instant and soon after. The learned judge thought so, and the medical assessor agreed, for it is clear, when his judgment is read, that he took the opinion of the assessor on the medical effect of the evidence in the light of his medical experience and knowledge, and judged of the legal effect for himself. It is quite true that every case must be proved, and something more is needed than a state of facts which is consistent with one view or the other. That something more is supplied if the facts show a probability one way or other. No one can frame a formula by which you can measure probabilities. We must judge in each case as we would in other affairs of life " (1) : See also *McArdle* v. *Swansea Harbour Trust* (2).

With due respect to the learned special magistrate it seems to me that he gave not enough weight to the facts of the case as distinct from the medical opinion. In an abstract way he endeavoured to reason from those opinions to a general conclusion of a scientific character, which he then applied to defeat the widow's claim. He said :—" You have an elderly man of sixty-three. Unbeknown to himself he was suffering from coronary sclerosis. Whether he had any previous trouble from his heart either at the time Dr. Covernton saw him or at any time I am unable to say. From the results of that disease in all probability sooner or later he would have died, whether waking or sleeping, working or idling. On the science of medicine as I find it on the evidence before me, death from coronary

(1) (1915) 8 B.W.C.C., at pp. 295, 296.
(2) (1915) 8 B.W.C.C. 489, at pp. 495, 496.

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thrombosis cannot generally be related to exertion, but that death in such cases is a mere happening in the chronological order of things. If death does occur concurrently with or closely connected in point of time with exertion, it cannot satisfactorily be said to have been caused by such exertion. In these circumstances, when I find death to be closely connected in point of time with exertion by Forst, I am unable, notwithstanding the suggestions of the experts called by the applicant to connect the two in such a way as to say the exertion caused the death. In my view I would be merely 'jumping to a conclusion' if I did so." The conclusion there expressed that death from coronary thrombosis "cannot generally be related to exertion" does not deny that death from that cause can ever be related to exertion. Indeed, there is substantial medical evidence in the present case that exertion can produce coronary thrombosis and that where there is danger of its supervening the patient should avoid physical exertion. It seems to me that the special magistrate has erected the guardedly advanced theory of some of the medical witnesses that coronary thrombosis is independent of exertion into the decisive presumption "that death in such cases is a mere happening in the chronological order of things." Such a presumption is not justified even by the evidence adduced to contradict that part of the medical evidence which tends to prove that coronary thrombosis may sometimes result from exertion.

The Supreme Court took a more balanced view and considered what was the proper inference to draw from the whole of the evidence, that is to say, the facts and circumstances of the case and the medical opinion. The substance of their conclusions is carefully and cautiously expressed in these terms:—"If the formation of the thrombosis is a gradual process, which may sometimes occupy several days before occlusion occurs, it follows that the activities of the patient immediately preceding the onset of the attack may be purely coincidental. But the evidence in this case is that the thrombus must have formed very quickly. We think that all the experts are in agreement upon the point that the clot must have formed after the deceased had been working on the jib although, as Professor Cleland points out, the antecedent changes might have

occupied several days. It seems to us that the effect of the evidence is clearly and impartially stated by the same witness in the following passages :—'In Forst's case he could not have done any severe exercise with that portion of the vessel nearly occluded or one would not expect him to' undertake 'much severe exercise and that is why I think that the final clot that killed him was formed rapidly immediately prior to his death . . . I remember the exertion that Forst had been engaged in. It is my opinion that the final clot formed after he came down from the crane. I think that as soon as the clot formed he would be unable to do anything. I can't see any special connection between the work done and the formation of the clot. At first sight one would naturally be inclined to associate any unusual effort with the fatal result. That is I would. I would try and see some reasonable association between the two but in the case of thrombosis in the coronary artery one's previous experiences being taken into consideration one fails to see an association that appeals to one as being right. If you hold your breath I understand that your pulse becomes weak. I understand that it would be expected to slow the circulation if the exercise done by Forst involved his holding his breath. That would facilitate clot formation. If the clot had been formed on the jib he would have fallen down. The slowing of the circulation tends to let the platelets drop out and it would facilitate the formation of the clot.' We agree with the witness that, speaking generally, it must be difficult and probably impossible to relate an attack of coronary thrombosis to any particular activity of the patient, and we concede that, even in this case, it is difficult to feel any great confidence in the inference, but it seems to us that we have here the coincidence of strenuous physical exertion occurring at or about the time when the thrombus must have commenced to precipitate. If the expert evidence shows, as we think it does, that physical effort is commonly—although not invariably—the inciting cause of that phenomenon, we think that we are entitled to draw the inference that Professor Cleland felt unable to draw. We fully accept his view that the evidence is in some measure inconclusive. For the purposes of a scientific deduction it may be insufficient, but we repeat that courts of justice are

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entitled and bound to act upon the probabilities of the case." The basis of these conclusions is well established by the evidence, but the decision of the court of first instance rests on an inadequate and somewhat abstract basis. I do not think it should be restored.

In my opinion the appeal should be dismissed.

Appeal dismissed with costs.

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C. C. B.